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Bacteremia Caused by CDC Group Ve-1 in Previously Healthy Patient with Granulomatous Hepatitis

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This is a case report of CDC group Ve-1 bacteremia in an otherwise healthy patient with granulomatous hepatitis.

CDC group Ve-1, a nonfermentative, gram-negative bacillus, was first described in 1928 and is an unusual isolate of questionable clinical significance. Recently, two cases of infection with this organism were reported in patients with debilitating underlying disease (1, 7). This is a report of CDC group Ve-1 bacteremia in a previously healthy patient with granulomatous hepatitis.

Case history. A 61-year-old male was admitted with a 6-week history of fatigue, low-grade temperature, generalized myalgia, and a 22-lb (ca. 10-kg) weight loss. About 1 month before admission, the patient was treated empirically with a 2-week course of oral erythromycin followed by a 2-week course of doxycycline with no clinical response. Laboratory evaluation at that time revealed a hemoglobin of 11.9 g/dl; a leukocyte count of 15,800/mm^3 with 13% band forms, 84% polymorphonuclear forms, and 7% lymphocytes; serum iron of 17 mg/dl; and an alkaline phosphatase level of 246 Bodansky units. The patient had no significant medical history. He had no allergies and was not taking any medications.

The physical examination at his admission was normal except for a temperature of 102.5°F (ca. 39.2°C) taken orally. The initial chest roentgenogram and urinalysis were normal. Urine culture and blood cultures were sterile. Complete blood count revealed a hemoglobin of 10.7 g/dl and a leukocyte count of 13,000/mm^3 with 5% band forms, 53% polymorphonuclear forms, 23% lymphocytes, and 13% monocytes. His rapid plasma reagin, rheumatoid factor, and antinuclear antibody tests were negative. The sera of the patient did not contain hepatitis B surface antigen, hepatitis B core antibody, or hepatitis B surface antigen antibody. His Coombs direct and indirect antibody tests were negative, and his cytomegalovirus immunoglobulin M titer was <1:10. His technetium bone and gallium scans were normal, as was an ultrasound of his abdomen. A computerized axial tomography scan of the abdomen and a liver-spleen sulfur colloid scan revealed nonhomogeneous uptake in the liver. His Westergren erythrocyte sedimentation rate was 45 mm. A bone marrow biopsy revealed a hypercellular marrow with active growth of all cell lines; special stains of the biopsy, as well as cultures for acid-fast bacilli and fungi, were negative. A percutaneous liver biopsy revealed sharply circumscribed, nonnecrotizing granulomas with very little fibrosis but with active inflammation in both the portal and hepatic spaces which was consistent with granulomatous hepatitis. Cultures of the liver biopsy specimens were subsequently negative.

The initial hospital course of the patient was characterized by daily temperature spikes of 102 to 103°F (ca. 38.9 to 39.4°C) orally. On day 10 of hospitalization, a gram-negative bacillus identified as CDC group Ve-1 was isolated from one of two aerobic blood cultures. The gram-negative bacillus grew on blood agar plates in rough-surfaced, yellow-pigmented, nonhemolytic colonies. It was catalase positive and oxidase negative and exhibited other chemical characteristics on the Vitek panel (Vitek Systems, Inc., Hazelwood, Mo.) and API 20E (Analytab Products, Plainview, N.Y.) from growth on blood agar plates (Table 1; 4). The organism was identified as CDC group Ve-1 on automated Vitek identification and API 20E reagent strips, and this identification was confirmed by the Centers for Disease Control, Atlanta, Ga. Antibiotic susceptibility testing by the automated Vitek method and by microdilution demonstrated susceptibility to amikacin, ampicillin, chloramphenicol, gentamicin, and tobramycin and resistance to cefoxitin.

Resolution of fever, myalgia, anorexia, and lethargy occurred after 4 days of antibiotic therapy. The leukocyte count and differential returned to normal, and the patient was discharged after 14 days of intravenous gentamicin and clindamycin. After 3 months, the patient had gained 5 lb (ca.

TABLE 1. Biochemical characteristics of group Ve-1 isolate

Test	Reaction
Urea	Negative
Arabinose	Positive
Citrate	Positive
Adonitol	Negative
Glucose	Positive
Maltose	Positive
Xylose	Positive
Arginine	Positive
Tryptophan	Negative
Raffinose	Negative
H_2S	Negative
Lysine	Negative
Bile esculin	Positive
Sorbitol	Negative
ONPG[a]	Positive
Lactose	Positive
Sucrose	Negative
Rhamnose	Negative
Gelatin	Positive
DP300	Negative
Acetate utilization	Negative

[a] ONPG, *o*-Nitrophenyl-β-D-galactopyranoside.

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2.2 kg), was afebrile, and had a very mild elevation of alkaline phosphatase to 143 Bodansky units. After 6 months, the patient was asymptomatic and afebrile, had gained 10 lb (ca. 4.5 kg), and had a normal complete blood count and liver function tests.

Discussion. CDC group Ve-1, previously classified as *Chromobacterium typhiflavum*, is a gram-negative bacillus for which the name *Pseudomonas luteola* has been proposed (5). Only 14 clinical isolates had been reported before 1970 (8), and clinical illness with the organism has been recognized only recently. The two reported clinical infections caused by group Ve-1 were prosthetic valve endocarditis with onset 2 weeks after valve replacement (7) and bacteremia and acute hemorrhagic pancreatitis in a patient receiving steroids for systemic lupus erythematosus (1).

The patient discussed in this report was previously healthy. He was found to have granulomatous hepatitis during his hospitalization. He did not have an intravenous catheter and had not had an invasive procedure at the time of the illness. This organism had never been isolated at our institution from any source. The rapid clinical response of the patient to appropriate antibiotics, as evidenced by a normalization of temperature and leukocyte count as well as a feeling of well-being, suggests that the blood isolate was a pathogen and not a contaminant. The antibiotic susceptibilities of the organism were similar to those previously reported: it was susceptible to ampicillin, ticarcillin, mezlocillin, gentamicin, tobramycin, and amikacin and resistant to cefoxitin and trimethoprim-sulfamethoxazole (1, 8). Resistance to ampicillin has been reported, although this strain was susceptible (3).

The source of the CDC group Ve-1 bacteremia and its relationship to granulomatous hepatitis are unclear. Infections with *Yersinia* and *Brucella* spp. and mycobacteria have been associated with granulomatous hepatitis (6, 9). There are no reports of nonfermentative gram-negative bacilli causing granulomatous hepatitis. The alkaline phosphatase level of the patient remained elevated for several months after antibiotic therapy and normalized 6 months later. It is possible that the granulomatous hepatitis was unrelated to the bacteremia (2). The source of the bacteremia is unknown. This case of CDC group Ve-1 bacteremia in a healthy patient with granulomatous hepatitis broadens the spectrum of disease caused by this organism.

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